Results: Conventional echocardiographic measurements (LV end diastolic diameter, LV end systolic diameter, LV EF) and LV volumes by 3 DE were similar between the groups. Interventricular septum and posterior wall thickness were increased, compared to controls. (1.27±0.07 cm to 1.1±0.19 cm, p=0.02; 1.25±0.23 cm to 0.9±0.02 cm, p=0.01, respectively). In TDI analysis, we observed marked reduction in LV peak systolic velocity (Sa) (0.06±0.008 m/s to 0.14±0.02 m/s, p=0.0001). LV longitudinal peak systolic strain (9.66±1.29% to 17.60±2.18%, p=0.0001) and strain rate (0.21±0.08 1/s to 1.66±0.56 1/s, p=0.0001) were significantly impaired in patients, compared to controls, demonstrating subclinical ventricular systolic dysfunction. Significant positive correlation was obtained between energy loss index and LV strain/strain rate. (r=0.481, p=0.015; r=0.596, p=0.002 respectively). Aortic valve area was also positively correlated with LV strain (r=0.422, p=0,036).
Conclusions: Patients with AS have evidence of subclinical LV systolic dysfunction, despite preserved EF. Changes in LV geometry are correlated to impairment in LV function. Strain imaging-based novel echocardiographic techniques may provide additional data for detecting early deterioration in systolic function in patients with AS.

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OP-008

The Long Term Incidence and Predictors of Radial Artery Occlusion Following a Transradial Coronary Procedure

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Objectives: Our aim was to establish the long term incidence of radial artery occlusion and investigate its predictors.
Background: Radial artery occlusion (RAO) is an infrequent complication of transradial coronary procedures (TRA). To our knowledge, there are no studies reporting the incidence and predictors of RAO in the late term following TRA.
Methods: This was a single center prospective study. A total of 409 consecutive patients undergoing their first TRA were recruited. Clinical and procedural data were all recorded. Doppler ultrasound examination was performed at the time of 6-15 months following the TRA.
Results: RAO was detected in 67 patients and 342 patients maintained radial artery patency (RAP). The overall RAO incidence was 16.4% at late term. Patients with RAO were younger than the patients with RAP (55.9±9.7 years versus 59.1±9.4 years, p=0,014). The incidence of RAO in hypertensive patients was 9.8%, lower (p<0.001) than the observed incidence (23.0%) in non-hypertensive patients. RAO group has higher rate (28%, p=0.027) of post-procedural access site pain. Regression analysis revealed that hypertension was negative while post-procedural access site pain was positive independent predictors for RAO. In addition the relative risk for RAO also increased significantly (p<0.001) when the ratio of sheath/artery diameter (S/A) was >1.
Conclusions: The present study reveals that the long term incidence of RAO is 16.4%. Hypertension, post-procedural access site pain and S/A ratio >1 are independent predictors of the long term incidence of RAO.

OP-009

Anxiety Score As a Risk Factor for Radial Artery Vasospasm During Radial Interventions: A Pilot Study

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Aim: Radial artery approach is an increasingly used technique. The most frequent complication of this approach is arm pain due to vasospasm. Studies about role of anxiety level on vasopasm are lacking, thus we sought to determine the role of anxiety level on radial artery spasm.
Method: A total of 82 patients who had an indication for coronary angiography were enrolled to study. Radial artery vasospasm was determined according to addressing five signs as follows: persistent forearm pain, pain response to catheter manipulation, pain response to sheath withdrawal, difficult catheter manipulation after being "trapped" by radial artery and considerable resistance on withdrawal of the sheath. Patients who had at least 2 of the 5 signs were diagnosed with clinical radial artery spasm. All patients were evaluated with Hamilton Anxiety Scale questionnaire in order to evaluate level of anxiety.
Results: The ratio of male to female was 45/36. Vasospasm was observed 19.1% of the patient population. The rate of vasospasm was 4.4% for men and 38.4% for women. Mean anxiety score of the whole study population was 14.0±7.9. The score was 17.6±7.3 for women and 11.1±7.2 for men. Anxiety score was significantly higher in women (p<0.001). Vasospasm was strongly correlated with female sex (p<0.001, R=0.43) and anxiety score (p=0.007, R=0.29). Female sex was associated with higher anxiety scores (p<0.001, R=0.43). However, age was associated with neither anxiety score nor vasospasm (p>0.05).
Conclusion: Higher anxiety scores and female sex are risk factors for radial artery vasospasm.

Table 1

	Male (n = 45)	Female (n = 36)	P value
Age (years)	55.9 ± 12	55.9 ± 10	NS
Anxiety score	11.1 ± 7.2	17.6 ± 7.3	<0.001
Vasospasm (%)	4.4	38.4	<0.001
Height (cm)	172.1 ± 7.4	162.5 ± 6.5	<0.001
Weight (kg)	87.2 ± 13.6	79.8 ± 14.4	NS
Body mass index (kg/m^2)	29.4 ± 4.3	30.2 ± 5.3	NS

Comparison of male and female patients who underwent radial coronary angiography

OP-010

Predictors of Microvascular Obstruction Assessed by the Index of Microcirculatory Resistance Following Primary Percutaneous Coronary Intervention for Acute ST-Elevation Myocardial Infarction

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Purpose: We aimed to investigate the predictors of microvascular obstruction (MVO) among clinical, electrocardiographic, laboratory and angiographic parameters available on admission in patients with acute ST-elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI).
Methods: Forty-nine patients treated successfully by pPCI were enrolled. On post-pPCI day 4 to 5, index of microcirculatory resistance (IMR) was measured with the use of a guidewire tipped with pressure and temperature sensors. MVO was defined as IMR above the mean value of 31 U.
Results: The mean IMR was 31.2±14.5 U. MVO was present in 23 (46.9%) patients. At univariate analysis, age >65 (p=0.012), pain to balloon time>180 min (p=0.012), ST segment resolution (STR) at postprocedural 90th min <70% (p=0.05), lesion length (p=0.04), BNP (p=0.03) and D-dimer (p=0.05) levels on admission were found to be associated with MVO. At multivariate analysis, pain to balloon time>180 min (Odds ratio (OR) 2.94, 95% Confidence Interval (CI) 1.54 – 50, p=0.025), STR<70% (OR 5.5, 95% CI 1.64 – 20, p=0.05), BNP level on admission (OR 1.029 per unit increase, 95% CI 1.002 – 1.057, p=0.035), and D-dimer level on admission (OR 1.11 per unit increase, 95% CI 1.016 – 1.212, p=0.05), were found to be independent predictors of MVO.
Conclusions: In addition to the well known predictors of MVO such as delayed time to reperfusion and incomplete STR, we showed that BNP and D-dimer levels on admission independently predict the presence of MVO in patients with STEMI treated with pPCI.

OP-011

Assessment of Silent Neuronal Injury Following Coronary Angiography and Intervention in Patients with Acute Coronary Syndrome

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Objectives: To date limited data is available regarding the occurrence and predictors of silent neuronal injury (SNI) after percutaneous coronary intervention (PCI). The aim of this study is to evaluate the incidence and predictors of SNI after coronary angiography and intervention by serial measurement of serum neuron specific enolase (NSE) in patients presented with acute coronary syndrome.
Methods: Ninety-eight consecutive patients presented with ACS who underwent coronary angiography and intervention were included in the study. NSE was studied before and 18 hour after the PCI. Clinical and echocardiographic characteristics were analyzed and independent predictors of SNI were evaluated.

Results: NSE levels significantly increased after coronary angiography and intervention compared to baseline levels (22.03±27.70, 10.08±3.15 consecutively). Baseline characteristics were similar between groups (Table-1). Left ventricular ejection fraction in the SNI+ group was significantly lower than SNI- group (43.71±12.51%, 50.84±9.34%, p=0.002). Maximal CK-MB, Troponin-I, Syntax Score (SS), of SNI+ group were significantly higher than SNI- group (103.83±99.22, 51.92±78.33, p=0.006; 50.04±66.18, 19.18±30.50, p=0.002; 103.83±99.22, 51.92±78.33, p=0.006 and 50.04±66.18, 19.18±30.50, p=0.002 successively). SS and performing PCI were the independent predictors of SNI (p=0.009, OR=1.06, 95%CI=1.014-1.107, p=0.036, OR=4.262, 95%CI=1.097-16.56).
Conclusions: PCI and coronary artery lesion complexity may increase the risk of SNI in patients with acute coronary syndrome.

Table-1

Variable	SNI+	SNI-	p value
Age, year	62.68±9.33	64.01±11.27	0.566
Male n(%)	26 (83.8%)	49 (73.1%)	0.243
Body Mass Index, kg/m2	28.06±4.03	28.01±4.85	0.515
Hypertension, n(%)	10 (32.3%)	25 (37.3%)	0.627
Diabetes, n(%)	10 (32.3%)	24 (35.8)	0.730
Hyperlipidemia, n(%)	9 (28.1%)	22 (32.8%)	0.782
Smoking, n(%)	11 (35.5%)	20 (29.9%9	0.752
Atrial fibrillation, n(%)	2 (6.5%)	8 (11.9%)	0.404
Mean Arterial Pressure, mmHg	100.71±13.80	98.26±12.14	0.374
Percutaneous Coronary Intervention, n(%)	21 (67.7%)	30 (44.8%)	0.034
SYNTAX score	28.13±14.68	18.71±13.23	0.002
Fluoroscopy Time, min	6.37±4.23	4.70±4.16	0.069
estimated Creatinine Clearance, ml/min	103.65±30.51	99.10±34.47	0.530
Glucose, mg/dl	127.71±60.40	133.07±67.80	0.707
LDL, mg/dl	141.32±42.59	135.19±47.74	0.733
Total Cholesterol, mg/dl	201.21±66.84	205.85±55.17	0.718
Hemoglobin, g/dl	14.42±1.59	13.97±1.70	0.210
Mean Platelet Volume, femtolitre	8.27±1.51	8.21±0.93	0.797
Red cell Distribution Width, %	13.55±0.91	13.36±1.42	0.498
CK-MB, ng/ml	103.83±99.22	51.92±78.33	0.006
Troponin-I, ng/ml	50.04±66.18	19.18±30.50	0.002
Left Atrial diameter, mm	38.29±4.09	38.51±5.85	0.853
Left Ventricular End Diastolic Diameter, mm	49.90±5.83	49.30±5.03	0.600
Left ventricular End Systolic Diameter, mm	36.10±5.76	33.39±6.00	0.037
Ejection Fraction, %	43.71±12.51	50.84±9.34	0.002
Asendan Aorta diameter, mm	34.71±4.11	34.57±4.20	0.875

OP-012

Treatment of Coronary No-Reflow With Intracoronary Vasodilators Added to Intravenous Tirofiban

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Objective: We prospectively assessed the management and short term prognosis of no-reflow phenomenon in a tertiary referral hospital.
Methods: We included 46 patients with ST-segment elevation acute myocardial infarction (STEMI) and occurrence of no-reflow phenomenon after primary percutaneous coronary intervention. They were all received intravenous tirofiban and then randomized into one of the 3 groups: intracoronary adenosine (n=16), intracoronary verapamil (n=15) or serum physiologic as placebo (n=15). Intracoronary drugs were administered after stent implantation. Thrombolysis In Myocardial Infarction (TIMI) frame counts were used to assess coronary flow.
Results: Groups were similar in terms of age, sex, STEMI localisation, diabetes, hypertension, mean reperfusion time. Hemotologic and biochemical blood parameters and procedural properties were also similar between groups (stent size and length, diameter and number of balloon(s) and stent(s) used). Verapamil plus tirofiban therapy had significant effect in restoring impaired coronary blood flow, decreasing TIMI frame count from 73±44 to 52±48 (p=0.024). However, adenosine and serum physiologic administration were not found to be so effective in decreasing TIMI frame count (from 81±35 to 71±46, p= 0.084; from 74±32 to 71±37, p=0.612; respectively). In-hospital and 6-month survival rates were similar among groups.
Conclusion: Tirofiban plus verapamil restored the impaired coronary blood flow more effectively than tirofiban plus adenosine and tirofiban only. Although both verapamil and adenosine have multiple effects on coronary circulation and even on platelet aggregation, verapamil as a calcium channel blocker, not only relieves microvascular spasm but also reduces calcium influx into ischemic cells, relieves cellular edema and restores calcium homeostasis. It may be hypothesized that these additional features put verapamil ahead of other drugs and may explain the better results we obtained with verapamil in comparison to adenosine.

OP-013

A Simple Index to Predict Adverse Clinical Outcome Associated with Acute Myocardial Infarction in Primary PCI Era

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Background: The major determinant of final infarct size for a given coronary occlusion is the size of myocardium that the artery perfuses. Defining the initial area-at-risk (AAR) for infarction has major clinical implications since it permits an accurate estimate of myocardial salvage provided by reperfusion therapies. We proposed a new index 'Relative Importance Index (RII)' to predict potential infarct size in patients with anterior MI.
Aim: The aim of the study is to assess the predictive role of RII for reduction in systolic function and its relation to adverse clinical outcomes.
Methods: One Hundred twenty-three acute anterior MI patients with their first acute coronary syndrome incident were consecutively and prospectively enrolled to the study. Patients with a clinical history of congestive heart failure, valvular heart disease, and previous coronary revascularization were excluded. All patients underwent primary percutaneous coronary intervention (PCI) for revascularization. Angiographic exclusion criteria were 1) pre-procedural TIMI flow ≥2 in the infarct related artery, 2) chronic total occlusion of other arterial territory 3) any visible collateral flow to infarct related artery 4) diffuse disease at proximal segments of coronary arteries that precludes defining reference segment. Coronary diameters were measured with quantitative coronary analysis program. RII was calculated by dividing culprit segment diameter to the sum of diameters of LAD, Cx, and RCA at their proximal segments (Figure 1). Troponin I (TnI) concentration at 72 hour was chosen as a serological estimate of infarct size. We evaluated 1-month follow up rates of major clinical endpoints (MCE), which is defined as death, non fatal MI, stroke, and new congestive heart failure. Left ventricular EF (LVEF) at 1st month was chosen as an index for systolic function.
Results: RII was significantly and negatively correlation LVEF (r=-0.65, p<0.001) (Figure 2). As RII of culprit lesion increased there was tendency to end up with lower EF. Likewise, RII was significantly correlated with 72 h TnI (r=0.48, p<0.001). Patients were dichotomized according to median value of RII (median RII=0.30) (Table 1). Supra-median RII was associated with lower EF and higher incidence of composite MACE. The mortality (12.9% vs. 6.6%), non-fatal MI (6.5% vs. 3.3%), and new CHF (12.9% vs. 3.3%) rate in supra-median RII group trend higher but they did not reach statistical significance. An RII >0.30 had a 88% sensitivity and 60% specificity (ROC area 0.82, p<0.001, CI [0.73-0.90]) for predicting severe LV dysfunction (LVEF <30%) (Figure 3).
Conclusion: A Simple index derived from coronary angiography at time of primary PCI can predict LV systolic function loss and adverse clinical outcome in patients with acute anterior myocardial infarction.